A CASE OF LANDRY'S PARALYSIS, WITH AUTOPSY.

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It is a matter of much regret to us that the clinical history of this case is meagre and imperfect and that the autopsy was incomplete; yet in spite of these drawbacks the case is one presenting features of great interest. We are aware that there is room for difference of opinion as to the diagnosis, yet the history and the clinical observations of Dr. Diller, who watched the progress of the case, to our minds, favor this diagnosis rather than any other, viewed from a clinical standpoint. If the diagnosis of Landry's paralysis be accepted the post-mortem findings must be considered as of great value as throwing light upon the obscure question of the morbid anatomy of this disease.

History of the case by Dr. Diller:

R. W., married woman, aged fifty-three years, admitted to St. Francis Hospital, Pittsburg, May 10, 1894, with loss of power in legs which had developed suddenly about ten days previously. This loss of power was not absolute, but constantly progressed, although with assistance she was able to stand as late as June 10th. One week after admission she began to lose power in the arms and control of the vesical and anal sphincters. This loss of power in arms and legs and over sphincters increased steadily and became absolute about two weeks before her death, which occurred on August 8, 1894. About a week or ten days before death bulbar symptoms developed rather rapidly, as evidenced by increasing difficulty in phonation and deglutition. Indeed, during the last few days she lived swallowing was attended with such danger that she was fed by the stomach-tube.

Mentally she seemed bright enough until after the bulbar symptoms developed, when her mind became somewhat clouded.

Pain, tactile, and heat and cold sense were, as determined by tests on several occasions, unaffected. Both knee-jerks were lost. There was some swelling of both knee-joints (probably due to old rheumatic trouble), and forced movements of them caused outcries of pain.

At the time of death there was no appreciable atrophy of any part of the muscular system. The electric reactions were normal. She developed no bedsores. No fever or pain at any time (except upon motion as noted). Death was due to respiratory failure. Clinically, the case was one of ascending motor paralysis. The successive involvement of legs, arms, and muscles of deglutition and phonation, while sensation and nutrition remained intact, indicated that the disease-process was a progressive ascending one, involving the motor apparatus of the cord and medulla and leaving uninjured the sensory and trophic apparatus.

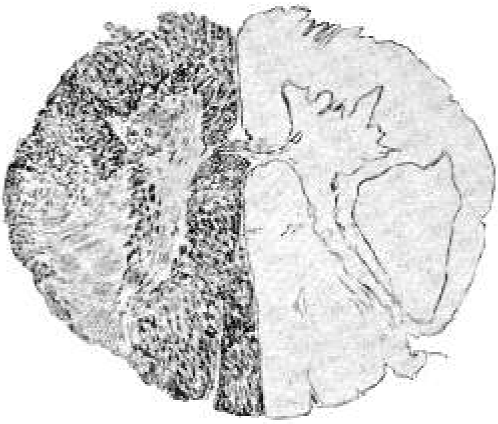
The absence of atrophy, of pains and of knee-jerks, and the preservation of sensation seemed sufficient to throw out the diagnosis of poliomyelitis, general myelitis, amyotrophic lateral sclerosis, and multiple neuritis.

From Landry's paralysis the case differs somewhat only in two features, viz., rather slow development and long course (fourteen weeks) and the paralysis of the sphincters. Cases are on record, however, in which the sphincters were involved.

Pathological Report and Analysis of a Case and of Literature,
by Dr. Meyer.

The spinal cord only and the anterior and posterior roots were examined. The stains used were Ehrlich-Biondi, hæmatoxylin, and Weigert-Pal. Macroscopically nothing could be seen in the cord that would have indicated disease.

FIG. 1.



Mid-cervical region.

The left side gives the relative appearance of the tracts of the cord, the right side the outlines of the degeneration in the lateral columns. Drawn with Edinger's projection apparatus; enlarged twelve times.

The sections, however, showed very plainly the following changes:

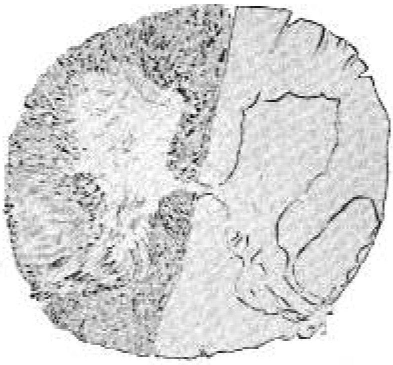
Weigert-Pal sections. In the cervical region (Fig. 1) the area of the pyramidal tracts is very distinctly outlined, decidedly lighter stained than even the anterior horns, though not so light as the gelatinous substance of the posterior horn. The direct cerebellar, antero-lateral, and posterior tracts are deeply stained; also the ground-bundles. Counterstain with carmine demonstrates the presence of a rather large amount of "connective tissue" in the region of the pyramidal tracts; in this tissue there remain a large number of medullated fibres well stained. The uncrossed tract is not affected. The same condition is noted in the upper and lower dorsal region and in the lumbar region. (Fig. 2.)

Biondi-Ehrlich stain. Marked hyperæmia of the area of the crossed pyramidal tracts and increase of neuroglia-fibrils. (Fig. 3.) The axis-cylinder of the pyramidal fibres slightly rarefied, but nowhere in a condition of decay or swelling. The process of disease is probably stationary in the cord.

Hæmatoxylin-stain shows a slight increase in the number of nuclei in the area affected.

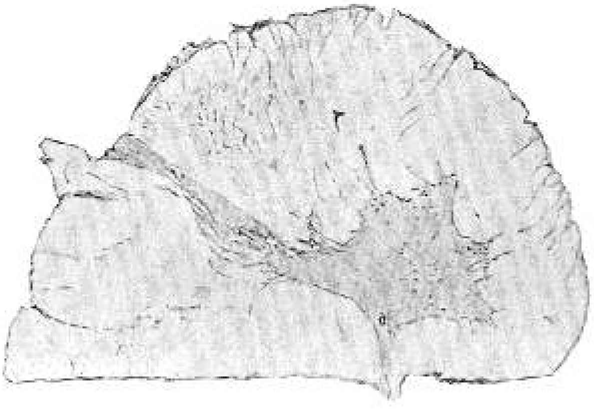
The anterior horns are not affected so as to give evidence of disease with the hardening in Müller's fluid. Only the marked pigmentation of the ganglion-cells attracts attention. The roots of the spinal nerves are free from degeneration. In the dorsal region defects can be noticed in the posterior columns. They are, however, only present in a piece of cord that was bent. This and the absence of reactive changes (increase of neuroglia-fibres) speak very much in favor of the view that the falling out of a number of axis-cylinders is a mere post-mortem injury.

FIG. 2.



Lumbar region.

FIG. 3.



Mid-cervical region.
Stain with Ehrlich-Biondi's color-mixture. Hyperæmia of pyramidal tract.

The findings are thus limited to a partial decrease of medullated fibres in the crossed pyramidal tracts, with secondary increase of the neuroglia-fibres, in the whole extent of the spinal cord, and excessive pigmentation of the anterior horn-cells.

The medulla oblongata and the brain, and also the peripheral nerves, were not available for examination.

A review of the cases of Landry's paralysis with autopsy was compiled by Dr. A. Cramer in 1892.[1] Several cases have been reported since.

A perusal of the literature up to the date mentioned shows that the clinical picture has been taken very broad, allowing quite a number of cases of plain polyneurites to be classified under the head of Landry's paralysis. Polyneuritis may be an acute ascending paralysis; but an acute ascending paralysis need not be Landry's paralysis because the disease shows first in the legs and then rises gradually to the upper segments of the body. The cardinal points for the differential diagnosis should be:

1. Flaccid paralysis of the muscle, spreading rapidly from one point over the rest of the body, generally beginning in the legs, but sometimes following the reverse order, as in the French zoölogist Cuvier.
2. Absence of muscular atrophy and of electrical reaction of degeneration.
3. Tendon and superficial reflexes absent.
4. Sensibility not, or only slightly, impaired.
5. Sphincters, as a rule, intact (exceptions rather frequent).
6. A criterion has been the absence of an anatomical lesion.

Among the twenty-seven cases with fairly satisfactory post-mortem examination, collected by Cramer, we find nine without findings, seven of which were reported before 1876. In the one of Kahler and Pick (1880) the peripheral nerves are not mentioned, although there was diminished electric reaction; the case of Strümpell (1883) is without examination of the peripheral nerves and does not contain any remarks on the electric reactions. Considering everything, there is some reason to believe that it is not pure chance that most of the cases without pathological findings date from a period when the technique was defective, and that all of them date from before the time when Weigert's hæmatoxylin method was published (1884); the case of Albu forming the only exception. (See below.)

The peripheral nerves alone were found diseased in six cases. The case of Eichhorst (1876) would now be classified clinically as polyneuritis (anæsthesia and reaction of degeneration in paralyzed parts). One case of Dejerine (1879), one of Eisenlohr (1890), might have been suspicious for polyneuritis (paræsthesia and diminished electric reaction). Among the three genuine cases that remain in this group, the one of Nauwerck and Barth (1889) is the most remarkable, since the patient, a girl of eighteen years, lived three months, with flaccid motor paralysis of legs, trunk, and arms, and finally died of bulbar symptoms. The sphincters were free, sensibility only slightly interfered with, electric reaction normal. Clinically the case resembles ours most of all.

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[1] Dr. Cramer: Die Pathologische Anatomie der Landry'schen Paralyse. Zusammenfassendes Referat. Cbl. f. allg. Path. u. path. Anat., Bd. iii. pp. 6–21.

In seven cases the spinal cord alone was affected. In only two of these (Eisenlohr, 1878, and Immerman, 1885) the peripheral nerves were positively pronounced intact; in the others the question remains unanswered. Baumgarten (1876) found bacteria and cell-infiltration in the cord; v. d. Velden (Leyden) disseminated myelitis, with cloudy swelling of anterior horn-cells; Eisenlohr (1878) exudates round the bloodvessels and numerous small hemorrhages; Kummel (1881) observed hemorrhages in the restiform bodies in a case of typhoid fever (fourth week), who died four days after the onset of paralysis, without disorder of the reflexes; Aufrecht (1881) had two cases with small myelitic foci. Immerman's case of six weeks' duration showed numerous granule-cells and hyaline degeneration of anterior horn-cells.

In the remaining five cases lesions of both peripheral nerves and spinal cord are reported. The earliest one dates from 1868 (G. Harley and J. Lockhart Clarke): softening in the anterior roots and in the cord; duration, six days. In the case of Schulz and Schultze (1882) reaction of degeneration and paralysis of sphincters are recorded. Decay of medullary sheaths in peripheral nerves, swelling of axis-cylinders in the cord, and cloudy swelling of motor ganglion-cells and also disintegration of the muscle-fibres were found; duration, four weeks. Hoffmann (1884) reports, in his case, swelling of axis-cylinders of anterior roots, lateral columns, pyramidal tracts, and corpora restiformia and capillary hemorrhages. Centanni's case (1889) showed invasion of peripheral nerves by bacilli and slight affection of cord without bacilli; Eisenlohr (1890) had one case with interstitial and parenchymatous neuritis and obtained three kinds of micro-organisms from the sciatic nerve and the spinal cord.

Seven autopsies of fairly typical cases have been published since, and are accessible to us in abstracts of the *Neurologisches Centralblatt.* In one of them (J. Watson, *British Medical Journal,* December 10, 1892) nothing was found but slight congestion of the membranes of the cord. It is impossible to say, from the abstract, how extensive an examination was made. The case of Pribykow (*Neurologisches Centralblatt,* 1894, p. 716) was one of polyneuritis, probably involving the spinal cord; he found numerous granules with Marchi's method, where other stains had not shown anything abnormal. The peripheral nerves were extensively affected. In the case of Albu (*Zeitschrift für klinische Medizin,* Bd. xxii.) the macroscopical and microscopical and bacteriological examinations were negative. The patient died on the fifth day.

Hun[1] published another typical case of twelve days' duration, with degeneration of fibres of the anterior roots of the cauda equina and thickening and infiltration of the walls of the anterior spinal vein; and Klebs ("Ueber Landry'sche Paralyse," von Prof. Klebs in Zürich, *Deutsche med. Wochenschrift,* 1891, No. 3) found in his case hyaline thrombi within the area of the central arteries of the spinal cord.

There remain the two cases of Hlava[2] and Eichberg.[3] They both showed round-cell infiltrations and small hemorrhages in the anterior horns of the spinal cord, without degeneration of the anterior roots.

Adding the findings of our own case to those mentioned above, we would sum up our view as follows:

Landry's paralysis is a symptom-complex without quite uniform post-mortem findings. There may be cases of very rapid course in which the naked eye, and even microscopical examinations with the help of older methods, does not reveal any lesion. With the improvement of the technique, these cases have become very rare, and there is reason enough to believe that the most recent technical proceedings would have demonstrated changes in most cases. Hereby one of the most important items that induced Landry to describe this symptom-group as a disease, *sui generis,* has fallen; the post-mortem findings can no longer be said to be negative, but they are at least not quite uniform.

Since polyneuritis and the forms of paralysis depending on it have been separated more closely from the so-called spinal paralysis, many cases apparently following the type of Landry's paralysis, but exhibiting sensory disturbances, electric reaction of degeneration, and perhaps the characteristic pain, spontaneous or on pressure, of neuritis, have led to the conclusion that every case was properly a polyneuritis, with or without spinal complication, but not far enough developed to give all the characteristic symptoms. This is the view of J. Ross and J. S. Bury,[4] who analyzed ninety-three cases of Landry's paralysis up to 1888, and claimed that it was too acute to give all the symptoms of polyneuritis, but identical with it, because it had the same etiology. Eisenlohr takes a broader view, on ground of his own cases with autopsy. He, and with him Cramer, states that either the peripheral nerves alone, or the peripheral nerves and the central nervous system, may be diseased, and that the peripheral nerves are involved in almost all cases. Clinical and anatomical evidence to this effect has been obtained in abundance.

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[1] Henry Hun: The Pathology of Acute Ascending Landry's Paralysis. New York Medical Journal, May 20, 1891.

[2] Polymyelitis Acuta Disseminata Partialiter Hæmorrhagica (Paralysis Landry). Archives Bohèmes de médecine, Tome IV., fasc. 2, p. 256.

[3] A Case of Acute Ascending Paralysis with Rapidly Fatal Issue. Medical Record, February, 1891.

[4] On Peripheral Neuritis, 1892.

While this paper was going through the press a patient presenting the following features was observed by one of us (Dr. Diller): A middle-aged man, known as a steady drinker, developed loss of power in legs, besides pain and great tenderness of nerve-trunks, atrophy, loss of knee-jerks, failure of memory, and other mental symptoms. Subsequently the trunk and then the arms became involved in the same manner as the legs. Finally, bulbar symptoms developed, and death soon followed. Paralysis of vesical and anal sphincters and bed-sores (which developed to enormous size) were prominent features after the trunk and arms had become involved. Clinically this would seem to be a case of acute ascending alcoholic multiple neuritis, in which there was serious involvement of the cord.

In looking over the history of the diseases of the nervous system that do not depend on a well-localized lesion, we find several instances of similar fluctuation of explanatory hypotheses. Paretic or paralytic dementia, for instance, has been styled leptomeningo-encephalitis chronica diffusa; now we know that the process of degeneration is not limited to the pia, nor to the cerebral cortex, nor to the brain as a whole, but that there are few cases in which the spinal cord and the peripheral nerves are not affected as well. The same holds for alcoholic neuritis. In a great number of the cases there are marked central disorders, especially loss of memory, delusions, etc.; in lead-poisoning, too, we find not only affections of the peripheral nerves, but also of the spinal cord and brain; nevertheless, we find both these forms simply classified as polyneuritis, because the peripheral lesion gives the best analyzed symptoms. We find also that the old division of peripheral and spinal and cerebral disease can no longer be upheld for anatomical reasons. A peripheral motor-fibre can never be a unit, since it is merely a process of a ganglion-cell in the anterior horn of the cord or in the motor nuclei of the medulla oblongata. The motor symptoms, and also the electric reaction, are the same whether the interruption of function takes place in the anterior horn-cells or in their processes. A lesion of the ganglion-cell involves the processes, and a lesion of the processes involves the ganglion-cell; hence it is practically impossible to outline the limitation of consecutive changes even to a well-localized affection of a nerve, as, for instance, a contusion or section, and certainly much less so the area involved by a diffused process of inflammation or degeneration, and it is certainly arbitrary to call the process simply myelitis or simply polyneuritis.

While we owe most of the present accuracy of clinical neurology to the rigid attempt at localization of lesions, localization must take a broader field and begin more and more to cover the localization of function and its anatomical substrata without paying attention to accidental topographical dividing-lines, if we wish to do justice both to the recent findings and to the traditions of clinical neurology.

To Moebius (*Abriss der Lehre von den Nervenkrankheiten,* Leipzig, Amb. Abel, 1893) is due the credit of having broken the orthodox method of classifying nervous diseases according to the localization of their lesions only. A hemorrhage, an injury, a tumor, a focus of localized inflammation, must, of course, determine symptoms that are best systematically arranged according to the old system depending on localization. But for a great proportion of diseases the etiological characteristics give a very satisfactory systematic basis, and there seems to be little doubt that ultimately the general pathology of the nervous system will be much benefited by the departure.

The entire group of intoxications, either of real toxic or of microbic origin, is characterized by only relatively localized affections of the nervous system. It is true that polyneuritis is most frequently the most prominent feature of the acute forms; but very often the symptoms cannot all be explained on the basis of merely peripheral disease, and pathological anatomy proves plainly that a purely peripheral disease in the old sense is hardly possible (compare the research of Nissl, *Cbl. f. Nervenheilkunde,* July, 1894), and that indeed there are, as a rule, microscopical changes not only in the large ganglion-cells of the anterior horns, but also more diffusely in other parts of the cord. It seems in none of the cases an accurate examination of the brain has been made. Probably there would be lesions there as well.

There is probably no writer who would to-day refuse the view that Landry's paralysis and allied acute destructive processes in the nervous system are most probably of toxic origin. The nature of the toxic matter and the special disposition of the individual exposed will determine the character and extent of the symptom-complex. What are they? There are four cases on record in which bacteria were found in the cord or in the peripheral nerves, or in both. Baumgarten (1876) found characteristic bacteria in the cord. Curschmann found typhoid bacilli in the cord of a case that died during typhoid fever. Centanni (1889) described bacteria in the peripheral nerves of his case, and Eisenlohr found no less than three kinds of micro-organisms in one of his cases. On the other hand, there are quite a number of cases in which very careful bacteriological examination of all the organs was quite negative. This seems to be certain that there is not a special germ that would alone and always produce the same effect, Landry's paralysis. If one of us (Dr. D.) submits the suggestion that the micro-organism that engenders the poison is one which is harmless in the body under ordinary circumstances, but which under peculiar circumstances becomes pathogenic, and results in bringing about this disease, we must admit that the cases in which a complete bacteriological examination of the internal organs was negative still remain unexplained; on the other hand, the cases following typhoid infection and influenza will probably be explained as caused by specific reaction to those specific infectious diseases. How far Edinger's Ersaztheorie will explain the peculiar distribution of the lesion in the individual cases remains to be shown when more attention will be paid to such questions of etiology and predisposition. His idea is that every toxic process properly is an interference with nutrition, amenable to repair within certain limits, but irreparable when the modulus of elasticity is surpassed; disorders of nutrition are most deleterious in those mechanisms that are least deeply rooted, least stable. It may satisfy many to know that in locomotor ataxia the post-syphilitic disorder of nutrition and function attacks almost exclusively the posterior columns of the cord and the posterior roots. But there arises the question: Why are certain spinal segments affected in preference to others (in cervical tabes, etc.), and why does the patient not show the symptoms of general paralysis, etc.? In a similar way we should ask, Why does the process of intoxication in our case affect largely the pyramidal tracts and perhaps the posterior columns and least the anterior roots, while other patients with the same affection have almost exclusively polyneuritis?

Our case does not give any clue in this direction as far as it could be examined. It seems to be unique, since no other report mentions a marked atrophy in the pyramidal tracts (cf., however, the case of Hoffman). There is one clinical case reported that might have an element of similarity if we would look upon it as one of a lesion within the possibilities of repair, involving the peripheral nerves, especially of the upper segments of the body (including the motor nerves of the eye), and the terminal apparatus of the central motor path (the end brushes the pyramidal tract), especially in the lumbar region. Muratow reported the case in the Society of Neurologists and Alienists at Moscow, January, 1892, as a case of acute ascending paralysis (Landry). (*Neurolog. Centralbl.*, 1892, p. 493). It is anomalous in many respects and an instance worth knowing of the polymorphous character of the disease. There was atrophic paralysis of the four extremities, the muscles of the trunk and the diaphragm, serious bulbar symptoms, and hyperæsthesia and pain in the course of the nerves, besides other purely neuritic symptoms. Moreover, there were nystagmus, dilatation of the right pupil, and exaggerated knee-jerk and ankle-clonus on both sides. The patient recovered.

If we recognize a certain uniformity in the *modus operandi* of the exciting causes of acute ascending paralysis, and if we recognize that the same cause may produce various types of disease in various individuals, we might look out for a common term for these forms, since the types are rarely pure. Oppenheim is evidently in favor of the extension of the term Landry's paralysis when he says that it had been too narrowly outlined by Landry. Eichberg (*Medical Record*, February, 1891) tries to avoid the difficulty by calling his case one of acute progressive motor paralysis. This name is certainly too narrow, since it does not take into account the hyperæsthesia that was present in his own patient. Leyden divides the cases into neuritic and bulbar ones, according to the starting-point and the chief distribution of the degenerative process. This classification is also too one-sided. The real bulbar cases are very rare as compared to those that begin in the lumbar cord, and it would be at least safe to make a third group of principally spinal cases.

There is a group of acute general nervous affections which have common features in their etiology, their rapid onset, and their course, most probably the result of acute infectious or toxic toxæmia or of exhaustion. According to the age and predisposition of the patient and the character of the poison, fairly definite groups of functional elements are involved which allow us to recognize marked clinical pictures with corresponding anatomical conditions.

1. In early childhood the prevalent forms are acute poliomyelitis; acute encephalitis is probably not belonging here, against the view of Strümpel.

2. In the adolescent and in the adult, polyneuritis, with distinct prevalence of the peripheral nerves, and Landry's paralysis, with prevalence of spinal symptoms.

There seems to be little doubt that acute "delirium grave" forms a parallel to these affections among mental diseases. These groups are by no means rigidly separated. Thus, we know of diphtheritic acute nerve-intoxication, that it comes into the picture of polyneuritis even in children; and in the adult the acute nerve-intoxication shows various transitions from polyneuritis to the spinal form, Landry's paralysis, and even involvement of the central motor path. The prevalence of the motor symptoms is very marked.

Our means of diagnosis are insufficient at present to localize the lesion in every clinical case more than approximately. The pathological findings vary according to the duration of the disease, and certainly also according to the methods employed for the examination.

More stress should be laid in the future on the study of etiological and predisposing factors, and, if possible, more elaborate precautions should be taken in the autopsy. On ground of the present experience the pathological examination should comprehend a study of at least the chief peripheral nerves, the cord, the medulla, and the cerebral cortex (at least the motor area) with the recent rapid stains (Nissl and Rosin) and with the osmic acid and ordinary fixation (Müller's fluid). Moreover, a bacteriological examination of the parts mentioned and of the internal organs should be made.[1]

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[1] The cases of Oettinger and Marinesco and Villa came to our notice since the above was written, and are in no way opposed to the views put forth.